MH88D0760

NATIVE AMERICAN
RESEARCH AND TRAINING CENTER

Monograph Series

DIABETES AND NATIVE AMERICANS:
SOCIO-CULTURAL CHANGE, STRESS AND COPING

Paul Skinner, Ph.D.
Dana Silverman-Peach, M.A.

October, 1989

THE UNIVERSITY OF ARIZONA
TUCSON, ARIZONA

MH88D0760

DIABETES AND NATIVE AMERICANS: SOCIO-CULTURAL CHANGE, STRESS AND COPING

Paul Skinner, Ph.D.
Dana Silverman-Peach, M.A.

October, 1989

This study was supported by a grant from the
National Institute on Disability and Rehabilitation Research
U. S. Department of Education
Grant #G0083094

TABLE OF CONTENTS

I.) Introduction....................................1

II.) Diabetes and Native Americans....................2

III.) The Nature of Diabetes...........................4

IV.) Diabetes, Hyperglycemia, and Stress...............5

A. Diabetes, Hyperglycemia, and Maladaptation to Stress....................................6

B. Empirical Evidence of the Relationship Between Stress, Hyperglycemia, and Diabetes.....8

V.) Socio-Cultural Change, Stress, Diabetes, and Native Americans....................................11

VI.) The Nature of Psychogenic Stress..................16

VII.) Stress and Coping Strategies.....................20

VIII.) Native American Coping Strategies and Diabetes........23

Appendix A.......................................26

Appendix B.......................................28

Acknowledgments..................................32

Bibliography.....................................33

ERRATA PAGE

Page 3	Line 7. Delete the word "second" and change reference to: (personal communication, Dr. Kermit Smith, 1990; Dr. Smith is the Phoenix Area Diabetes Program Coordinator for IHS).
Page 5	Second paragraph, first line should read: "The pathophysiology of" type II diabetes is ...."
Page 7	Last paragraph, first sentence should read: In theory, then, the pathophysiology of diabetes may be viewed as the effect of an "imbalance" (influenced directly or indirectly by chronic stress) in the systemic mechanisms that regulate metabolism.
Page 14	Last paragraph, second to last line: change insulin to hypoglycemics.
Page 24	First paragraph, last sentence should read: "How then can this information be used to recognize and utilize effective coping strategies by Native Americans who have diabetes or who are at risk for diabetes?

Diabetes And Native Americans: Socio-cultural Change, Stress And Coping

Paul Skinner, Ph.D.

Dana Silverman-Peach, M.A.

I.) INTRODUCTION

Among the factors that usually receive serious concern in the etiology and control of non-insulin dependent diabetes mellitus (NIDDM), that of psychogenic stress is usually ignored. In spite of the considerable research over the years showing that stress is a key factor for diabetics in their day-to-day efforts to maintain stable blood glucose levels, consideration of the effects of stress is ignored in recommended compliance strategies for the control of hyperglycemia, the primary diabetic symptom, which is the primary goal of all proposed interventions. Unfortunately compliance with recommended medical treatment, including use of oral hypoglycemics, special nutritional programs, exercise programs, and weight loss, does not guarantee control or avoidance of complications.

There is increasing interest in the effect of such factors as stress, socio-cultural change, and attitude on blood glucose levels in diabetics both because these factors are known to influence metabolism and blood glucose control and because these factors influence the individual's responsiveness to recommendations for compliance (Surwit and Feinglos, 1988; Harris, Linn, and Pollack, 1984; Peyrot and MacMurray, 1985). Thus

TABLE OF CONTENTS

I.) Introduction..1

II.) Diabetes and Native Americans..........................2

III.) The Nature of Diabetes..................................4

IV.) Diabetes, Hyperglycemia, and Stress....................5

  A. Diabetes, Hyperglycemia, and Maladaptation to Stress..6

  B. Empirical Evidence of the Relationship Between Stress, Hyperglycemia, and Diabetes.....8

V.) Socio-Cultural Change, Stress, Diabetes, and Native Americans....................................11

VI.) The Nature of Psychogenic Stress.....................16

VII.) Stress and Coping Strategies.........................20

VIII.) Native American Coping Strategies and Diabetes........23

Appendix A...26

Appendix B...28

Acknowledgments....................................32

Bibliography.......................................33

campaign have significantly increased life-expectancy and coping skills for diabetics, the incidence and prevalence of this chronic disease continues to rise, particularly within Native American communities. The effects of diabetes on the general population are profound, but its impact on the Native American minority is much greater. Although it was once rare in Indian communities, type II non-insulin dependent diabetes mellitus (NIDDM) is now a common affliction, and diabetes is currently the second leading cause of outpatient visits to Indian Health Service (IHS) facilities (IHS, 1985). Brosseau, Bata, and Marquart (1984) point out that in the American Indian population of over 1.4 million, one person in three is at risk for type II diabetes. In comparison, only one person in forty-two in the general population is at risk for NIDDM.

The incidence of NIDDM varies significantly among tribes. The Pimas of southern Arizona have an incidence of NIDDM of about 50% in the adult population over age 35 (Sievers and Fisher, 1984). Health officials estimate that 50% to 70% of all Tohono O'Odham adults over age 35 will develop the disease (Hoffman and Haskell, 1984). The incidence of NIDDM is over 20% in eleven other tribes and over 10% in fourteen additional tribes. This contrasts with an overall diagnosed incidence of NIDDM in the population of the United States of 2.35% for all age groups (Sievers and Fisher, 1985).

The disabling and life-threatening secondary effects of diabetes similarly affect the Indian population with a far greater impact than in the general population. During the 1984 fiscal reporting period for the IHS hospital on the Tohono O'Odham reservation, 21 amputations were reported - an increase in amputations of at least 32% over the previous fiscal year. This represents an expectation of almost two amputations per month in a population of approximately 7,331 (personal communication,

Supervisor of Community Health Nurses, IHS Hospital, Sells, Arizona).

Conditions relevant to compliance with standard medical treatment for Indian diabetics are very different from those experienced by diabetic patients living in more positive socioeconomic conditions and more conveniently developed surroundings. Yet even in these "positive" conditions, compliance among diabetics in mainstream society, particularly with regard to diet, ranges only from 10-35% (Rosenstock, 1985). Given the phenomenal incidence and prevalence of diabetes among Indians, the relative deficit in preventative and restorative care available to Indian diabetics represents a critical and urgent need and offers a compelling challenge for the development of innovative and effective interventions that could benefit the general population as well.

III.) THE NATURE OF DIABETES

Because of its profound physical, social, and economic effects, diabetes among Native Americans has become the subject of intense study during the last two decades. However, as knowledge regarding diabetes has increased, so has the diversity of opinion regarding its clinical definition and pathophysiology, and the appropriate intervention to reduce secondary and tertiary complications. No longer considered a single disease entity, type II diabetes is now thought to be a constellation of related pathologies, all of which ultimately result in abnormally high blood glucose levels (Notkins, 1979).

The precise biochemical failure that leads to a diabetic state appears to be due to two related processes: diminished insulin action (insulin resistance), and a defect in the conversion of glucose to glycogen within the cell (Bogardus, 1987). Under normal circumstances, a high concentration of glucose in the blood stream results in the pancreatic

release of insulin in order to facilitate the uptake of the glucose through the cell membrane (insulin binds with the cell membrane, enabling the cell to absorb glucose), where it is biochemically converted to glycogen. Under stress (e.g., exercise), the glycogen stored in the cell is converted back to glucose to be used as an energy source by the cell in carrying out its normal metabolic functions.

Type II diabetes is initially characterized by hyperinsulinemia, i.e., the quantity of insulin and proinsulin in the blood stream is higher than normal, in part because of the high concentration of glucose in the blood. For reasons that are still not clearly understood, the insulin does not bind to the cell receptor (insulin resistance), and thus the glucose does not enter the cell, but remains at high levels in the blood, triggering further secretion of insulin.* Because of the lack of a supply of glucose for energy requirements, the cell becomes energy starved (diabetics frequently report extreme hunger and thirst) although there is a high concentration of glucose in the blood (hyperglycemia).

IV.) DIABETES, HYPERGLYCEMIA, AND STRESS

Diabetes, as well as other chronic diseases and disorders, has traditionally been viewed from a symptomatic perspective. As a result, attention has focused on the development of specialized treatments to ameliorate the symptoms of the disease; thus, the biopsychosocial context (psychological, social, and environmental factors) in which diabetes arises and must be controlled has largely been ignored. More recently,

---

*See Temple et al (1989), who states that type II diabetics have high concentrations of proinsulin in their blood stream, not insulin. Radioimmune assays for insulin do not distinguish between insulin and proinsulin. Proinsulin does not bind to the cell receptor, resulting in hyperglycemia.

considerable evidence has emerged regarding psychophysiological (mind-body) interactions in neuroendocrine function in metabolism, demonstrating the effect of psychogenic stress and coping in the development and course of diabetes (Surwit and Feinglos, 1988). These findings have greatly enhanced the interest in the biopsychosocial investigation of the prevention and treatment of diabetes.

As a result of this research, some experts have begun to view diabetes as a neuroendocrine-mediated metabolic imbalance which is heavily influenced by pituitary-adrenal-cortical activity, and therefore highly sensitive to psychogenic stress (Hinkle, Evans, and Wolfe, 1951; Bradley, 1979; Vague, et. al., 1979; Surwit, Feinglos, and Scovern, 1983).* These researchers have concluded that in response to stress the endocrine function, regulated by the central (CNS) and autonomic nervous systems (ANS), is a significant factor in fluctuations of diabetic control for both type I and type II diabetics and may be implicated in the etiology of Type II diabetes. For example, a diabetic who is in control and does not require insulin may suddenly require insulin as a result of grieving over the death of the spouse, a potentially very stressful experience. This need for insulin occurs because during the experience of stress the cells have an increased need for energy. This additional "stress" has therefore exacerbated the diabetic state.

A. Diabetes, Hyperglycemia, And Maladaptation To Stress

Many researchers have described diabetes as part of a maladaptive

---

*Emphasis on central nervous system (CNS) and autonomic nervous system (ANS) mediation of neuroendocrine and metabolic processes provides a useful theoretical framework to explore the role of psychogenic stress and related affective states in the etiology and course of chronic disease in general and diabetes in particular (See Appendix A for a discussion of the interaction between central and autonomic nervous systems in stress, and see Appendix B for a discussion of the neuroendocrine function in stress).

response to chronic stress (Hinkle and Wolfe, 1952; Surwit and Feinglos, 1988). For example, Hinkle and Wolfe (1952) described diabetes as "a bodily adaptive (actually maladaptive) pattern integrated at a high level of brain activity but inappropriate to meet a crisis." In other words, they defined diabetes as a stress related disease. How stress actually may lead to diabetes is not clear. It is well known in sensory physiology that receptor cells that are overstimulated (i.e., stressed) as a function of the frequency of stimulation (rate of occurrence), stimulus intensity, and stimulus duration lose sensitivity to normal stimulation temporarily or permanently, i.e., the cells demonstrate temporary and permanent threshold shifts in sensitivity. Furthermore, such overstimulation of receptor cells results in exhaustion of the cell and may culminate in cell destruction.

One of the major causes of cell exhaustion is loss of potassium ions. As cells lose more and more potassium, they function less and less effectively, and finally start to die. Also the loss of potassium ions results in significant shifts in cell plasma pH balance and leads to poor cell metabolism (Tortora and Anagnostakos, 1984).

Thus it could be hypothesized that frequent or prolonged intensive hormonal and biochemical stimulation of the pancreas could produce a maladaptive shift in the sensitivity threshold of the insulin-producing cells of the pancreas and could also impair and exhaust peripheral cells (e.g. muscle cells), thus impairing normal metabolic function. Such a maladaptive shift in cell function and related metabolic dysfunction could be involved as a causal mechanism in diabetes.

In theory, then, the symptomatology of diabetes may be viewed as the effect of an "imbalance" (resulting directly or indirectly from chronic stress) in the systemic mechanisms that regulate metabolism. Thus the diabetic individual may be characterized as one whose natural, adaptive

physiological feedback mechanisms have become maladaptive. Over a prolonged period of time (diabetes is a disease that develops slowly), reactive, chronic stress may lead to the third phase of the General Adaptation Syndrome, which has been described by Seyle (1973) as exhaustion. This exhaustion of peripheral cells may precipitate or exacerbate a diabetic state.

B. Empirical Evidence of The Relationship Between Stress, Hyperglycemia, And Diabetes

Surwit et al. (1985) have demonstrated in animal experiments that stress affects blood glucose levels in type II diabetes and that hyperglycemia can be conditioned in a Pavlovian manner. Using classical conditioning techniques on a species of obese mice "susceptible to type II diabetes," the authors demonstrated that hyperglycemia can be induced as a result of "environmental stress." Two groups of obese mice with comparable blood sugar levels were placed on similar diets; one group was subjected to environmental stress (physical constraint), which resulted in elevated blood sugar levels (close to 400 mg/dl) and reduced insulin secretion. Comparing "diabetic" to non-diabetic mice under stress (using a forward and backward moving platform), the authors found that the diabetic mice showed elevated blood sugar levels (about 350 mg/dl) while the non-diabetic mice showed much smaller increases in blood sugar levels (about 180 mg/dl).

Additional experiments were then conducted in a classical conditioning paradigm in which a metronome was started every time the mice were subjected to the stress of a moving platform (Surwit et al., 1988). The mice with hyperglycemia began showing increased blood sugar levels (over 250 mg/dl) as soon as the ticking began—even before the platform moved. Conversely, the ticking did not produce elevated blood sugar levels in the

non-diabetic mice (Surwit et al., 1988). This later finding is most remarkable because it indicates that generalization of physical and psychological stress can occur from direct (moving platform plus metronome) to associated stimuli (metronome only) and can be sufficient to produce hyperglycemic effects.

Surwit et al. (1985) have also demonstrated that the hyperglycemic effect under stress could be reduced by giving the mice a tranquilizer before exposing them to the stressful stimulation. One group of mice was given benzodiazepine prior to exposure to environmental stress (physical constraint); the control group was not given the tranquilizer. During the experience of stress, the tranquilized animals showed lower blood sugar levels than the non-tranquilized control group. The authors concluded that type II diabetes can be induced by stressful events and ameliorated by stress reduction through the use of tranquilizers such as the benzodiazepines.

Given these results in animal experiments, Surwit and Feinglos (1986) then sought to evaluate the effects of stress on blood glucose levels in diabetic patients. Nine female and three male patients with type II diabetes in poor control who were not using insulin were chosen because they demonstrated variation in diabetes control as a consequence of stressful events resulting from hospitalization. Six of the 12 patients were placed in an experimental group and were taught EMG-feedback assisted relaxation training in a hospital setting that allowed for control of diet and physical activity. The other six patients were designated as the control group and were exposed to identical conditions of diet and physical activity, but did not receive relaxation training. After 9 days, the experimental group showed significantly lower blood glucose levels based on oral glucose tolerance testing. Conversely patients in the control group

had significant increases in blood glucose levels. The results of this study suggest the possibility that some diabetics can be taught to use relaxation techniques to lower their blood glucose and maintain better glycemic control.

One of the implications of this study is that control of diabetic symptomatology may be amenable to behavioral regulation of autonomic nervous system activity, relaxation therapy, and biofeedback training, underscoring the apparent influence of stress on the blood glucose levels of diabetic patients. In a related study conducted at UCLA that also demonstrated that relaxation could help lower blood sugar levels, subjects over a six week period were taught to identify the physical symptoms of stress and to use relaxation techniques to reduce their body's response to the stress. The same subjects completed another six week period in which no stress reduction techniques were used. The subjects showed lower blood sugar levels when they used relaxation techniques compared to when they did not.

One interesting finding from these studies is that relaxation therapy was found to be useful to type II diabetics only. Similar experiments with subjects with type I diabetes did not result in comparable results (Surwit and Feinglos, 1988).

Feldberg, Pyke, and Stubbs (1985) provided further support that stress plays a significant role in type II diabetes by demonstrating that a large dose of morphine given intravenously raises blood glucose 100-200% and that this effect continues for about 8 hours. The same result occurs if adrenaline is slowly infused intravenously, a process which is comparable to a prolonged sympathetic discharge in response to stress. The authors concluded that such a mechanism may in some circumstances be implicated in clinical syndromes such as non-insulin dependent diabetes. Furthermore,

Vermes et al. (1985) reported that the beta-endorphin and ACTH levels were 3-4 times higher in NIDDM patients than in a control group. In this regard, the opioid peptide hypothesis of repression predicts that repressive coping is associated with increased functional endorphin levels in the brain, hyperglycemia, and a suppressed immune response. A study by Jamner, Schwartz, and Leigh (1988) confirms this hypothesis. The authors demonstrated that repressive personality types as measured by the Marlowe Crowne Social Desirability and Taylor Manifest Anxiety scales were mildly hyperglycemic and also had depressed immune responses. However, the relationship between increased opioid peptide levels and hyperglycemia is still unclear. McCubbin et al. (1987) suggest that opioid peptides may actually attenuate the hyperglycemic effects of sympathetic nervous system activity.

V.) SOCIO-CULTURAL CHANGE, STRESS, DIABETES, AND NATIVE AMERICANS

The Native American population of the United States has survived despite extraordinary physical, psychological, social, and cultural suppression over the past several centuries, and the stresses from this suppression are still very much a part of daily life for thousands of urban and reservation Indians. This suppression and resultant stress have contributed to dramatic changes in the family structure and lifestyle and have also contributed greatly to the severe economic and health problems suffered by this population.

Four major factors that characterize the nature of stress experienced by these Native Americans have been described by Joe and Miller (1987): 1) Native Americans of the United States have been involuntarily uprooted from ancestral lands that they perceived as central to their sense of well being and identity; 2) These Native people have experienced disintegration of

traditional cultural and social values, resulting in profound psychological conflicts within families and communities; 3) These Native people have experienced usurpation of personal and tribal freedom and authority, and have become "powerless and dependent upon the federal government and impersonal institutions," resulting in a "learned helplessness and dependency;" and 4) many are poor, unmotivated, unskilled, and culturally unprepared for the competitive lifestyle (educationally and economically) that characterizes mainstream society. Life style for Native Americans has deteriorated under these circumstances from purposeful activities such as hunting and farming, which are of benefit to family and community, to one of sedentary dependency.

Diabetes was virtually unknown among American Indians prior to 1940 (West, 1977). One must therefore consider changing conditions since that time in the epidemiology of this rapidly expanding and pervasive disease. Certainly the problem of diabetes among Native Americans has been exacerbated by environmental and lifestyle factors such as isolation, abject poverty, massive unemployment, sedentary behavior or inactivity, poor living conditions, cultural barriers, and resultant chronic stress. Over-consumption of alcoholic beverages is commonplace, and as reported by Justice (1985), diet has changed from natural and high fiber protein foods to processed and high caloric foods. Moreover, the inaccessibility and insufficiency of health services renders the effective treatment of diabetes in Indian communities extremely difficult.*

---

*Standard medical compliance regimen for the treatment of diabetes usually includes significant life-style changes which involve strict dietary management, self-monitoring of glucose levels through frequent urinalysis and blood analysis (frequently on a daily basis, sometimes several times per day), self-administration of insulin injections or oral hypoglycemics, and careful attention to foot hygiene.

The following case histories demonstrate the nature of stress on an individual basis as perceived within the context of Native American culture and serve to suggest a relationship between stress and diabetes:

1.) An Indian woman experienced a series of events that were psychologically traumatic for her. First, her spouse, who was very devoted to her, suffered a diabetic coma and died unexpectedly, and she experienced a deep sense of loss and grief. Shortly after this event, her village was flooded when a dam shattered and numerous people lost their lives. Also she lost extensive Indian relics and artwork that could never be replaced. She experienced a profound sense of despair and entered into a deep depression. Her lifestyle prior to these events had involved heavy drinking and eating and a rather sedentary existence; however, this woman had never been diagnosed as diabetic nor did she have any awareness of diabetic symptoms. Yet in her deep and prolonged depression she experienced extreme fatigue; she started to feel "jittery," shaky, dizzy, and faint, experienced persistent hunger but felt too weak to eat regularly, experienced constant thirst and kept a huge plastic glass of liquid always in reach, experienced polyuria, and began losing a significant amount of weight. Finally as a result of the coercion of a friend, she went to see a physician who diagnosed her as diabetic. Remarkably, she was delighted with her diagnosis of diabetes because she felt that diabetes, unlike her experience of depression, was a problem that she believed she could deal with. As a result she overcame her deep depression by focusing her attention on her diabetes, and she was able to bring her diabetes and related symptoms under control.

Three years later, however, she lost a dear friend who died of cancer, and her blood sugar levels again went out of control. She reexperienced all her previous symptoms and had great difficulty bringing them under

control. Within six months, she lost a second very dear friend who died of a chronic disease, and she reexperienced all the symptoms that characterize a lack of glycemic control. Eventually as a result of dietary control, a modified exercise program, and the care of friends and loved ones, she was able to bring her diabetes under control again. She now describes her diabetes as a "wolf in a cage" that mounts a full attack in times of acute or chronic stress or if she does not maintain strict medical compliance.

2.) A relatively young urban Indian man lost his job and was unable to find employment, and as a result he could not support his wife and two very young children. His wife and children moved back to the reservation to live with her family, but he could not return with her because there was no hope of finding employment on the reservation. He experienced stress in the form of a great sense of guilt and bitterness, a feeling of unworthiness, and a profound sense of loneliness as he continued to search unsuccessfully for employment. He began to experience weakness and fatigue, irritability, and insomnia. He started to drink and eat compulsively, and he experienced constant thirst, hunger, and polyuria. Unexpectedly he was recalled to his former job, which necessitated a physical examination, and he was diagnosed as diabetic.

3.) A middle-aged diabetic Indian woman who works in Tucson in an Indian program had maintained very effective glucose control by means of diet and exercise. Within a day of learning of the sudden death of her father, she experienced loss of energy, dizziness, and faintness so severely that she had to be driven home from work. Her blood sugar was greatly elevated, and she experienced many of the symptoms of hyperglycemia. She spent several days under a strict medical regimen using oral insulin before she was able to bring her blood sugar and other symptoms back under control.

4.) A middle-aged Indian man with teenage children was abandoned by his wife who left him for another man. The abandoned husband was greatly depressed, and he felt a deep sense of loss and shame that he was unable to cope with. He experienced chronic stress and depression, and began to feel physically weak and tired so that he often felt unable to go to work. He visited an IHS clinic and was diagnosed as prediabetic and was put on insulin temporarily, although he privately declined to use the prescription. In time he started to adapt psychologically to his situation through family support, and with careful attention to rest and diet he slowly recuperated. He met another woman through his work, and they developed a close personal relationship although they did not marry. He has had no recurrence of symptoms of hyperglycemia and maintains normal blood sugar without the use of oral hypoglycemics.

The purpose of these examples is two fold: (1) to call attention to how abrupt lifestyle changes and extreme stress affects blood glucose levels, and (2) to point out the importance of positive coping strategies as a part of the intervention to resolve the problems of stress and significantly enhance glucose control.

While the effect of stress on blood glucose control in diabetics has been described (Surwit and Feinglos, 1988), the effect of stress upon the onset of diabetes is less clear. Anecdotally one often hears that diabetes was diagnosed after an episode of extreme psychological trauma and acute stress, or after a period of prolonged severe chronic stress. Systematic studies of this phenomenon are extremely difficult, however, because there is rarely "before and after" data, i.e., subjects rarely have taken a blood glucose test before the onset of the stressful period in their lives, therefore implicating stress more directly as a factor in the inset of the disease process.

VI.) THE NATURE OF PSYCHOGENIC STRESS

It is important to acknowledge that normal metabolic regulatory functions are both directly and indirectly affected by states of acute and chronic psychogenic stress. As described in this paper, acute and chronic psychogenic stress are considered to be parallels of the alarm and resistance phases delineated in Seyle's model of the General Adaptation Syndrome (Seyle, 1956; 1973; 1979; 1980).

The acute stress response described by Walter Cannon (1914) as the Fight or Flight Response (or the alarm phase) of the General Adaptation Syndrome is a useful physiological mechanism that allows humans to quickly mobilize physical resources in order to respond to sudden or unwanted, unexpected, or overtly dangerous stimuli or situations that are perceived as threatening. Such emergencies are rare. Occasionally individuals may also find themselves in situations which, while not overtly dangerous, are nevertheless cognitively appraised by them as being threatening or beyond their perceived abilities to cope, and which therefore also produce an acute stress response (Lazarus, 1984).

Normally when a potentially threatening situation in the environment is removed or the perception of threat passes, the acute experience of the physical stress response also passes and the individual returns to normal physiological function (homeostasis). This process is also described as normal adaptation. But if the experience is cognitively encoded and held in memory in the form of associations, the experience of stress does not extinguish or disappear, but remains as a reactive program (classical conditioning) which is readily restimulated by associated thoughts or perceptions, thus acting as an internal stimulus to the autonomic nervous system.

Thus what may have once been experienced as a direct and immediate perception of threat (causing an acute stress response) may now be represented in a symbolic and generalized cognitive schema which manifests as thoughts and ideas of threatening associations. This is psychogenic stress, i.e., stress which is caused by unconscious associations, memories, interpretations of external events as threatening based on prior experiences. For example, consider a young man who was physically abused by his father as a child, who on return visits to his parents and home is constantly reminded of traumatic experiences from his childhood. As a result, the young man reexperiences conflict, fear, and chronic stress before and after these visits. Moreover, these repeated stressful reactions are reinforced each time he returns home and may be generalized by association to related situations in his daily life--away from home or family—that lead to the same recurring, chronic stress response he experiences when he is visiting his parents.

The chronic psychogenic stress response can be best characterized as the secondary or resistance phase of the General Adaptation Syndrome. Thus the cognitive processes of the central nervous system in the young man described above are the generating factor in a chronic psychogenic stress response, and are therefore a key link in creating neuroendocrine imbalances that lead to maladaptive metabolic patterns.

Psychogenic stress responses are a result of unconsciously learned cognitive patterns (ways of thinking) that are generalized by association and lead to reactions that become apparent only through emotional and physiological manifestations, and thus are commonly misperceived as being caused by "external stressors." It must be understood that these effects of stress are but the symptoms; they are not the cause. The experience of stress is actually a direct result of the perception (interpretation) of

the external events based upon prior learning (conditioning) and belief; psychogenic stress is not caused directly by the external event itself. For example, the death of a loved one, often cited as a major "stressor," may be experienced as stressful (in the case of a sudden, unexpected death) or as a release from stress (in the case of extended chronic illness), depending upon the perception of the observer. The incident of the death is not in itself the cause of the stress; the cause of the stress is the perception or interpretation of the event by the observer. Thus psychogenic stress may be correctly understood as a cognitive event or decision arising from one's perception of a change in conditions. Furthermore psychogenic stress as a cognitively mediated event or decision becomes manifest through very specific biochemical/biological processes with measurable consequences.

Stressful cognitive events are commonly generalized through association (as in the example of the young man described above) and thus can be unconscious reactions. Indeed, stress commonly can be experienced physiologically and behaviorally without conscious awareness of the underlying internal or cognitive causation. For example, one may have observed a loved one suffer traumatically and die as a result of the secondary complications of diabetes. Thereafter, this observer might experience fear around medical facilities and health care providers, yet not be fully aware that he is making an association between the observation of the suffering and trauma of the loved one with diabetes and the sights, sounds, and odors of the medical facility. The observer may only be aware of feeling faint and terrorized if something reminds him of diabetes or of hospitals. Now consider what happens if sometime later this person is diagnosed as having diabetes. He may feel frightened, angry, or guilty because of the previous situation, and thus deny that he has such a problem

rather than confront this perceived threatening situation. Any unconscious reminder results in a stressful reaction that results in fear, cellular energy consumption, and an increase in blood sugar and insulin.

Thus, the "difference" between psychogenic and physiologic stress actually reduces to a cause and effect relationship. They are not two distinct and separate events. A cognitive event or decision may be causal and the resulting emotional and physiological events are the effect of this cognitive experience. The result is a shift in biological homeostasis, and if maintained or frequently restimulated, metabolic shifts occur which may lead to or exacerbate a hyperglycemic condition and diabetes. The resulting experience is called maladaptive coping.

Consider the following example. Two different care providers in a nursing home were assigned alternating shifts to care for a particularly difficult and demanding man who had suffered a stroke and who subsequently manifested dementia. One health care provider, despite the man's frequently obnoxious behavior, was reminded of his father and was particularly compassionate and loving toward this man. The other health care provider felt threatened and overwhelmed by the patient's behavior and literally developed a quivering lip and hypertension over several months during the relationship. The patient experienced a second stroke and died very suddenly. Care provider one experienced a sense of loss and a period of sadness. Care provider two was greatly relieved, and both his quivering lip and hypertension disappeared within a week after the patient's death. Clearly one's experience is not based on the form or conditions of a situation; rather, one's experience is based on one's perception (interpretation) and attitude towards a situation.

VII.) STRESS AND COPING STRATEGIES

Relating stress to diabetes and to Native Americans is complicated by two factors: (1) the nature of stress is commonly not understood, and (2) different socio-cultural values and beliefs lead to different perceptions of what may be stressful. In this regard, non-Indian health care providers often misperceive the stoic demeanor of Indian people (in the presence of non-Indians) as indicating the absence of stress. Nevertheless the same kind of events may be perceived as stressful across very different cultures, e.g., the death of a loved one. Conversely, even within the same culture what may be stressful to one person may not be stressful to another.

An event itself does not necessarily cause stress. Stress is a result of perception or interpretation of the event. And in fact stress is not really the problem; the problem is maladaptive coping. As mentioned above, the death of a loved one may be perceived as an experience of loss and grief to one person and a blessing and relief to another. Thus whether an event or an experience is stressful is entirely dependent on the perception and reaction of the observer.

There exists a variety of techniques and treatments to reduce stress, including relaxation training and biofeedback, exercise, meditation, and Indian ceremonial activities such as traditional sweats, dances, and chanting. Moreover, improved dietary habits and physical and biomedical treatment including tranquilizing drugs can reduce psychogenic as well as biologic stress because such treatments, if responsibly used, reduce anxiety as well as a number of physiological symptoms. These methods, if used appropriately, may help the patient cope with stress and diabetes.

It is important to note, however, that stress reduction activities as well as other interventions, however critical the need, can have only

temporary and intermittent benefits. This is important to understand because stress reduction, per se, in the absence of significant cognitive or attitudinal change is treatment only--it does not solve the problem. Thus it is recommended that attitudinal change (i.e., change in perceptions, beliefs, and attitudes as the basis for stress) must accompany stress reduction and other forms of treatment for these activities to be truly effective (Skinner, 1989). This recommendation warrants explanation.

Successful stress reduction, regardless of the form, may well alleviate symptoms temporarily, but stress reduction does not provide persistent or stable benefits because it does not deal with the cause of the problem, i.e., our perceptions and attitudes. For this reason successful stress reduction methods are only tentative. Adaptive coping with stress may require various forms of treatment, but it must also include the releasing or the re-framing and re-interpreting one's cognitive experience. The only way to learn to cope positively with stressful conditions is to be willing to change one's perceptions and attitudes. Only in this way can we restore "balance" and "harmony" in one's life.

The critical issue then is to address the matter of positive coping or adaptation. The more we accept control or responsibility for our experience of emotional upsets and resultant stress, the more we can experience control of our lives and learn how to cope positively (maintain "balance"). The basic issue is our willingness and ability to assume responsibility for what we experience rather than to blame others; otherwise we seem to have no control over our lives. Ironically, denial of responsibility is the basis of our strongest resistance and the basis for chronic or persistent stress. We may or may not be in control of the conditions of our life, but we cannot avoid the responsibility for what and how we perceive, feel, and behave, and therefore what we experience.

We may unconsciously deny this responsibility, or we may repress or deny the conditions, but we do have to live with our decisions. To accept full responsibility for our experience is rarely an easy matter and virtually impossible to achieve without conscious re-learning. To fail to do so, however, is disastrous.

Denial of responsibility is a process we have "learned" and "used" all our lives as a defense mechanism. The way it works is as follows: We perceive and react "unconsciously" by interpreting the cause of our experience as being external to us (external stressors such as a diagnosis of diabetes), and as a result we feel powerless ("it's not my fault"--"Why me God?"). Because we are likely to assign guilt or blame to someone or something else outside ourselves, we emerge as helpless and hopeless victims, powerless to do anything about our situation. In so doing we deny responsibility and thus end up becoming a victim of our own defenses. As a result, we become entrapped by our own emotional reaction, which leads to inner conflict, fear, and stress.

To learn to live productively with the experience of stress, we must learn to change the way we perceive and to change our attitude. Only in this way can we release our upsets, our perceived threats and attacks, and our recurring or chronic stress. If we don't release the stress and conflict in this way, we become part of the problem instead of part of the solution; that is, we maladapt.

Positive coping requires a conscious relearning that results in a change in our perception and our attitude (Skinner, 1989). The process involves learning that actually neither the stress nor the diabetes is really the problem. The problem is our attitude towards it. Our attitude will determine our experience of the stress and how we cope with our diabetes. One can live a productive life while having diabetes, or one can

experience diabetes as a destructive disease. Changing one's attitude may seem simplistic; however, this attitudinal change is not merely "positive thinking." Attitudinal change requires that we open our minds about our concept of our "self," and about our values and beliefs, including our beliefs about disease and health. (Skinner, 1989).

All of us have stressful life experiences, and it is unrealistic to think that we can or even should try to avoid or reduce all stressful experiences. In fact stress can be perceived as either a problem or an opportunity. Stress is neither inherently good nor bad; on the contrary, the problem arises in the way we cope with the stress. We often cannot control the stressful conditions of our life, but we can and do control, consciously or unconsciously, our perceptions and how we experience these stressful conditions, i.e., how we cope (Skinner, 1989).

VIII.) NATIVE AMERICAN COPING STRATEGIES AND DIABETES

Despite the "white man's" conviction that the cause and "cure" of diabetes will be found through biomedical science and intervention, American Indians widely believe that cause and cure are inherently linked to cultural change, i.e., the loss and restoration of harmony and "wholism," respectively (Hagey, 1988). More specifically, studies by Garro (1988), Hagey (1988), and Tom-Orme (1988) provide extensive evidence that many Indian people believe that diabetes is a result of imbalances inflicted by the "white man" on Indian lifestyle, environment, and diet, and as a result, the majority of Indian people no longer live in harmony with themselves, each other, or nature. Indeed these socio-cultural changes, as experienced by Indian peoples, have been psychologically devastating. While Indians are commonly viewed by non-Indians as stoic and fatalistic peoples, the lifestyle for most American Indians has become

profoundly and pervasively that of conflict, fear, and chronic stress.

Compelling evidence has been presented that clearly demonstrates significant relationships among cultural change, stress, and diabetes. How then can this information be used to recognize and utilize effective coping strategies by Native Americans who have or at risk for diabetes?

Again, it is important to emphasize that stress is not the problem; the problem is maladaptive coping. Positive coping given stressful circumstances can lead to a productive life. Clearly the imbalance or disharmony that now engulfs Indian peoples as a result of cultural change, conflict, and stress, and specifically the rapidly expanding incidence and prevalence of diabetes, is not going to go away in the immediate future. Thus the challenge that faces Indian peoples (and non-Indian cultures as well) is to restore inner harmony in the presence of disharmony or conflict, fear, and stress. This challenge is not an easy one. It demands one's maximum human potential (Skinner, 1989), and yet it is perhaps the only alternative that is consistent with Native American goals and beliefs about the cause and treatment of diabetes as well as the many other major problems faced by Indian peoples.

Perhaps the most hopeful note for Native Americans, and the most valid and effective coping strategy about the cause and cure of many devastating so-called "white man diseases" such as diabetes, is to learn to cope with conflict and stress by revitalizing traditional Indian values and beliefs about the nature of harmony and how to maintain it for oneself. One of the most profound and pervasive pan-Indian goals and beliefs is that one can learn to live with external disharmony and still achieve and experience spirit/mind/body harmony within, and share that experience with others as the pathway of healing (Locust, 1985). This goal can be achieved through the practice of traditional Indian ceremonies, and through programs on

attitudinal change (Skinner, 1989).

The cultural experience of spirit/mind/body harmony is potent medicine that can restore inner peace, self esteem, and relationships (leading to reduction of bodily symptoms such as hyperglycemia and hypertension), empowering one to live a happy and productive life regardless of external conditions. This most important part of Indian medicine is desperately needed by and can be shared with all peoples regardless of tradition or culture.

APPENDIX A

Central And Autonomic Nervous System Functioning: The Mediators Between the External World and Physiological Response In Stress

In order to understand the connection between psychological stress and the physiological stress response, the basis for central nervous system control and regulation of other physiological systems must be clarified. One link that is often overlooked in understanding these interactions is the central nervous system (largely the interaction of the cerebral cortex, the reticular activating system, and the limbic system) which processes information into perceptual programs that provide interpretation of external events and the mechanisms for the emotional, physiological, and behavioral actions of the organism.

The central nervous system is a critical interface between the external environment and internal physiological systems. Furthermore, the central nervous system interprets and synthesizes information vital for the regulation of all internal systems. It organizes, stores, interprets, and integrates sensory information about both internal and external worlds, and mediates physiological responses through the autonomic nervous system.

The autonomic nervous system is functionally dependent upon the central nervous system, particularly the cerebral cortex and the hypothalamus (Tortora and Anagnostakos, 1984). Thus central nervous system activity both informs and regulates autonomic functioning on behalf of the perceived needs of the total organism (human body).

The central nervous system processes information, and through the

cognitive processes of selective perception, i.e., association, learning, and memory, enables the individual to interpret and give "meaning" to external events. In this way emotional, physiological, and behavioral responses are generated, consciously or unconsciously, which ultimately modify the body's internal systems, particularly the neuroendocrine system and various metabolic pathways that initiate shifts in biological homeostasis. If these responses are maintained for an extended period of time or are frequently restimulated (as a result of chronic stress or maladaptive coping), metabolic shifts may occur that produce a hyperglycemic condition and exacerbate diabetes.

APPENDIX B

Neuroendocrine Function In Stress

It is important to understand the interaction of the chemical regulators that are involved in maintaining metabolic homeostasis and to see how they function as feedback mechanisms. The mechanism of insulin action is based upon a homeostatic feedback system. Following the ingestion of a meal, carbohydrates are converted into blood glucose, which circulates in the blood stream. A rise in blood glucose level directly stimulates the beta cells in the pancreas to produce insulin, the principal action of which is to accelerate transport of glucose from the blood into cells and to accelerate conversion of glucose into glycogen for storage in the liver (glycogenesis). Insulin also reduces circulating blood glucose by inhibiting the conversion of stored glycogen into circulating glucose (glycogenolysis) and by slowing the conversion of other metabolic nutrients into glucose (gluconeogenesis). A drop in the blood glucose level following insulin action can also be sensed by the beta cells, which then reduce their insulin production.

Other hormones can directly affect insulin production, and in fact, any hormonal activity which causes a rise in the level of blood glucose will similarly increase the secretion of insulin by the beta cells of the pancreas. Any hormonal activity which causes a drop in blood glucose level will reduce the secretion of insulin by the beta cells (Tortora and Anagnostakos, 1984).

Glucagon is a product of the alpha cells in the pancreas. Its principal physiological activity is to increase blood glucose levels. Glucagon acts in opposition to insulin by accelerating the conversion of glycogen in the liver into glucose (glycogenolysis) and the conversion of other nutrients, such as amino acids, into glucose (gluconeogenesis). In

response to these processes, the blood glucose level rises. As with insulin, glucagon operates in a feedback loop in which low blood sugar levels are sensed directly by the alpha cells, which are then stimulated to increase glucagon secretion. High blood glucose levels are sensed by the alpha cells and result in decreased secretion of glucagon. Any metabolic change which causes a drop in blood glucose will stimulate the alpha cells to produce glucagon to counteract the low blood glucose level (Tortora and Anagnostakos, 1984).

The same sympathetic nervous system mechanisms and hormones which regulate metabolism also produce energy mobilization in response to stressful events. The stress response is mediated by CNS activity and CNS/ANS interaction in metabolism through hypothalamic, pituitary, and adrenal-cortical metabolic pathways (Tortora and Anagnostakos, 1984).

As indicated, the pancreas contains alpha and beta receptors that secrete glucagon and insulin, respectively, in carbohydrate metabolism. Both receptors are directly stimulated by different divisions of the autonomic nervous system and thus the pancreas also is under autonomic control. Stimulation of the beta adrenergic receptors through the sympathetic system facilitates insulin secretion; stimulation of the alpha adrenergic receptors through the parasympathetic system inhibits insulin secretion. In this way ANS stimulation may modulate insulin secretion in the normal regulation of the metabolism of carbohydrates (Williams and Porte, 1974).

Insulin and glucagon operate with mutual dependency as part of the same feedback interaction in order to maintain an appropriate range of homeostatic balance in the metabolism of carbohydrates. The ingestion of nutrient, however, is not the only factor that influences the dynamic interaction between insulin and glucagon in the regulation of metabolism

and associated homeostasis.

Other powerful hormones, described below, have hyperglycemic effects which can intrude on the feedback cycle and exaggerate the range of its fluctuations with pathological results. In particular, the hormones which are released during both the alarm and resistance phase of the General Adaptation Syndrome in response to stress can cause a transient rise in blood glucose which would effect both insulin production and effectiveness (Marble et al., 1985). This is a normal response to stress.

The catecholamines are stress hormones that have a significant effect on blood glucose levels. Synthesized by the adrenal medulla and produced in response to the increased sympathetic nervous system activity which accompanies the alarm phase of the General Adaptation Syndrome, the catecholamines cause blood glucose levels to rise very quickly, but only for a short period of time. These hormones (epinephrine and norepinephrine) stimulate the conversion of glycogen into glucose in the liver (glycogenolysis) and facilitate its release into the bloodstream. It is important to note, however, that both the sympathetic arousal of the alarm phase of the stress response and low blood sugar levels are signals which increase the production of the catecholamines (Tortora and Anagnostakos, 1984).

The increase in sympathetic activity which typifies the initial alarm response of the General Adaptation Syndrome is superseded by the secondary phase of the stress response: the resistance reaction (Seyle, 1956; 1973; 1979; 1980). The resistance phase of the stress response is designed to allow the body to continue responding to the challenge of perceived stressors long after the sympathetic arousal of the alarm phase has subsided (Tortora and Anagnostakos, 1984). This is a long-term physiological reaction during which the hypothalamus indirectly stimulates

the production of other important hormones, the glucocorticoids.

Synthesized by the adrenal cortex, the principal action of the glucocorticoids is to regulate normal metabolism and the long-term response to perceived stressors. They are produced more slowly and have a longer-lasting and more subtle effect on blood glucose levels than do the catecholamines, and thus have a more sustained effect on metabolic interactions. Cortisol, the most important of the glucocorticoids, increases the conversion of nutrients other than carbohydrates (such as amino acids) into glucose (gluconeogenesis). As in the case of catecholamine secretion, glucocorticoid secretion is stimulated both by low blood glucose levels as well as the hypothalamic stimulation which characterizes the resistance phase of the stress response. Thus, the catecholamines also may have the effect of increasing the feedback effect on the overall metabolic process (Tortora and Anagnostakos, 1984).

Finally, pituitary growth hormone strongly affects the feedback mechanisms of glucose metabolism. The production of growth hormone is increased in response to the hypothalamic stimulation that characterizes the resistance phase of the General Adaptation Syndrome. Growth hormone stimulates adipose tissue to release fats and accelerates the rate at which glycogen in the liver is converted to glucose and released into the blood (glycogenolysis). Since cells are using fats for energy under this circumstance, the result is an overall increase in the level of blood glucose that contributes to a hyperglycemic effect. The mechanism of growth hormone release is based upon a feedback loop like that operating in the other hormones discussed above. Again, both low levels of blood glucose and the hypothalamic stimulation of the resistance phase of the stress response are feedback signals that mediate the release of growth hormone (Tortora and Anagnostakos, 1984).

ACKNOWLEDGMENTS

Special appreciation is extended to Dr. Robert Young of the NARTC staff for identification of numerous references that provide scientific validation for several theoretical aspects of this monograph, and for his conscientious and persistent editorial and creative assistance in the development of this monograph.

BIBLIOGRAPHY

Bogardus, Clifton. (1987). Skeletal Muscle and Insulin Action in vivo in man. Current Concepts. Kalamazoo, MI.: Upjohn Co.

Bradley, Clare. (1979). Life events and the control of diabetes mellitus. Journal of Psychosomatic Research, 23:159.

Brosseau, James D., Bata, Bonnie R., and Kathleen Marquart. (1984). A Quiet Epidemic. Diabetes Forecast, 37:38-40, 62.

Cannon, W.B. (1914). The emergency function of the adrenal medulla for pain and in the major emotion. Am Journal of Physiology, Vol. 33:356.

Diabetes in America. (1985). National Diabetes Data Group: U.S. Dept. of Health and Human Services.

Feldberg, W., D.A. Pyke, and W.A. Stubbs. (1985). On the origin of non-insulin dependent diabetes. Lancet, 1:1263-64.

Garro, Linda. Cultural knowledge about diabetes. In: Diabetes in the Canadian Native Population: Bicultural Perspectives (ed. T. Kue Young). Toronto: Canadian Diabetes Association, pp. 97-110.

Hagey, Rebecca. Rationality of responses among Native American diabetes. In: Diabetes in the Canadian Native Population: Bicultural Perspectives (ed. T. Kue Young). Toronto: Canadian Diabetes Association, pp. 111-122.

Harris, Rachel, Margaret W. Linn and Linda Pollack. (1984). Relationship between Health Beliefs and Psychological Variables in Diabetic Patients. British Journal of Med Psych, 57:253.

Hinkle, Lawrence E., F.M. Evans, and Stewart Wolf. (1951). Studies in Diabetes Mellitus: IV. Psychosomatic Medicine, 13:160.

Hinkle, Lawrence E., and Stewart Wolf. (1952). The effects of stressful life situations on the concentration of blood glucose in diabetic and non-diabetic humans. Diabetes, 1:283.

Hoffman, B.H., and A.J. Haskell. (1984). The Papago Indians: historical, social, and medical perspectives. Mt. Sinai J. Med., 6:707.

Indian Health Service: A Comprehensive Health Care Problem for American Indians and Alaska Natives. (1985). Rockville, Md.: U.S. Department of Health and Human Services.

Jamner, Larry D., Schwartz, Gary E., and Hoyle Leigh. (1988). The relationship between repressive defensive coping styles and monocyte, eosinophile, and serum glucose levels: Support for; the opioid hypothesis of repression. Psychosomatic Medicine, 50:567-575.

Joe, Jennie R., and Dorothy Miller. (1988). American Indian Cultural Perspectives on Disability. Monograph. University of Arizona: Native American Research and Training Center.

Justice, James, M.D. (1985). Type II diabetes mellitus in the Tohono O'odham. Lecture given at O.R.D. in Tucson, Arizona, December 12, 1985.

Lazarus, Richard S., and Susan Folkman. (1984). Stress, Appraisal and Coping. New York: Springer Publishing.

Locust, Carol. (1985). American Indian Beliefs Concerning Health and Unwellness. Monograph. University of Arizona: Native American Research and Training Center.

Marble, Alexander, Leo P. Krael, Robert F. Bradley, A. Richard Christlieb, and J. Stuart Soeldner. (1985). Joslin's Diabetes Mellitus (12th ed.). Philadelphia: Lea & Febinger.

McCubbin, J.A., R.S. Surwit, C.M. Kuhn, C. Cochran, and M.N. Feinglos. (1987). Effect of opiate antagonism on stress-induced hyperglycemia in obese mice. Diabetes, 36, Suppl. 1.

Notkins, Abner Louis. (1979). The causes of diabetes. Scientific American, 241:62.

Peyrot, Mark and McMurray, James F. (1985). Psychosocial Factors in Diabetes Control: Adjustment of Insulin Treated Adults. Psychosomatic Medicine, 47(6):542.

Rosenstock, I.M. (1985). Understanding and enhancing patient compliance with diabetic regimens. Diabetes Care, 8(6):610-16.

Seyle, Hans. (1956). The Stress of Life. New York: McGraw Hill.

Seyle, Hans. (1973). The evolution of the stress concept. American Scientist, 61:692.

Seyle, Hans. (1979). The stress concept and some of its implications. IN Vernon and David M. Warburton, Ed., Human Stress and Cognition. New York: John Wiley and Sons.

Seyle, Hans (ed.). (1980). Seyle's Guide to Stress Research, v.l. New York: Van Nostrand Reinhold Company.

Sievers, Maurice L., and Jeffrey R. Fisher. (1985). Chapter XI. Diabetes in North American Indians. In: Diabetes in America. U.S. Department of Health and Human Services. NIH Publication no. 85-1468.

Skinner, Paul. (1989). Attitudinal Healing. (Book in Progress).

Surwit, Richard S., and Mark Feinglos. (1986). The effects of relaxation on glucose tolerance in non-insulin-dependent diabetes. Diabetes Care, 6:176.

Surwit, Richard S., and Mark Feinglos. (1988). Stress and autonomic nervous system in type II diabetes: A Hypothesis. Diabetes Care, 2:83-85.

Surwit, Richard S., Mark N. Feinglos, and Albert W. Scovern. (1983). Diabetes and behavior: a paradigm for health psychology. American Psychologist, 38:255.

Surwit, Richard S., James A. McCubbin, Elizabeth G. Livingston, and Mark Feinglos. (1985). Classically conditioned hyperglycemia in the obese mouse. Psychosomatic Medicine, 47:565.

Temple, Rosemary, Luzio, Stephen, Schneider, Annerose, Carrington, Christine, Owens, David R., and Wendy J. Sobey. (1989). Insulin Deficiency in Non-insulin Dependent Diabetes. Lancet, 1:293-95.

Tom-Orme, Lillian. (1988). Diabetes in a Navajo Community: A Qualitative Study of Health/Illness Beliefs and Practices. Ph.D. Dissertation, College of Nursing, University of Utah.

Tortora, Gerard J. and Nicholas P. Anagnostakos. (1984). Principles of Anatomy and Physiology. 4th Edition. New York: Harper & Row.

Vague, J., Combes, M., Tramon, M., Angeletti, P. H. Rubin, Hachem, A., Perey, D., Lansada, C.H., Ziras, Ramahandridona G., Jouve, R., Sambuc, R., and J. Jubelin. (1979). Clinical features of diabetogenic obesity. In J. Vague, P. H. Vague, and F.J.G. Eblin, (eds.), Diabetes and Obesity. Excerpta Medica. Amsterdam, pp. 127-47.

Vermes, I., E. Steinmetz, J. Schoorl, Van der Veen, E., and F. Tilders. (1985). Increased plasma levels of immunoreactive B-endorphin and corticotropin. Lancet, 2:725-26.

West, K.M. (1978). Diabetes in American Indians. Adv Metab Disord., 9:29-48.

Williams, R.H., and D. Porte, Jr. (1974). "The Pancreas": in Textbook of Endocrinology. 5th ed. Philadelphia: Saunders.